

Invited Perspective: Prenatal PM_{2.5} Exposure Associated with Adverse Birth Outcomes Requiring Medical Interventions

Marnie F. Hazlehurst^{1,2}  and Sheela Sathyanarayana^{2,3}

¹Department of Environmental & Occupational Health Sciences, University of Washington, Seattle, Washington, USA

²Seattle Children's Research Institute, Seattle, Washington, USA

³Department of Pediatrics, University of Washington School of Medicine, Seattle, Washington, USA

<https://doi.org/10.1289/EHP14290>

Refers to <https://doi.org/10.1289/EHP12880>

Air pollution is a threat to human health, especially for vulnerable populations, with effects that span the life course. In particular, accumulating evidence links air pollution exposure during pregnancy with adverse birth outcomes, such as low birth weight and preterm birth.^{1,2} These conditions put children at higher risk for illness in infancy and a range of health conditions into adulthood, including intellectual and developmental disabilities, obesity, diabetes, and heart disease.^{3–5} Pollutant exposures during pregnancy are also associated with adverse respiratory outcomes in childhood, such as asthma.^{6,7}

In this issue of *Environmental Health Perspectives*, Johnson et al. report results from their study of respiratory distress in a sample of >1,000 mother–child dyads across 10 Canadian cities, using air pollution exposures estimated from ground-level monitoring, satellite models, and land-use regression.⁸ They did not observe associations between air pollution and mild signs of birth distress, but they did find associations with severe newborn respiratory distress. Specifically, higher exposure to fine particulate matter [PM ≤ 2.5 μm in aerodynamic diameter (PM_{2.5})] was associated with increased risk of the need for assisted ventilation, administration of systemic antibiotics, and use of multiple clinical interventions for respiratory distress.

This work provides a novel contribution to the growing epidemiologic evidence for the adverse effects of prenatal exposures to PM_{2.5}, given that few studies have examined this outcome previously despite respiratory distress being a leading cause of neonatal morbidity and mortality worldwide.^{9–11} These outcomes reflect critical neonatal conditions that are only partially explained by known risk factors, but little attention has been paid to environmental etiologic agents.

Common risk factors for severe respiratory distress include birth trauma, infection, and preterm birth (the newborn population in the new study was restricted to term births).¹² Birth trauma usually leads to low Apgar scores immediately after birth, but Johnson et al. did not observe associations between higher air pollution exposure and low Apgar scores at 1 min after birth. Mechanisms for the harmful health effects of air pollution include inflammation, oxidative stress, and epigenetic programming, leading to disruptions in fetal development and immune dysregulation.⁶ Both epidemiologic and animal studies

have identified alterations in immune system markers with higher prenatal air pollutant exposures.^{13,14} The findings from the study by Johnson et al. further suggest that maternal PM_{2.5} exposure could increase the potential for bacterial infection in newborns requiring treatment with antibiotics. This could also be explained by preemptive treatment for infection by physicians when an infant has respiratory distress.

Normal development and function of the maternal–fetal–placental unit is critical for normal fetal growth and birth. Johnson et al. also investigated several maternal factors, including prepregnancy metabolic disorders and hospitalization during pregnancy, finding that underlying maternal conditions, as well as maternal health during pregnancy, may amplify the adverse effect of PM_{2.5} exposure on newborn health outcomes. Although additional work is needed to confirm these findings in studies with large sample sizes, these data suggest that clinicians should be aware that environmental factors may have significant impacts on newborn outcomes, requiring medical interventions.

This study contributes to the growing literature on prenatal exposure to PM_{2.5} and child health, with implications for both policy and practice. Associations were observed in this study at pollutant levels—median PM_{2.5} concentration of 8.9 $\mu\text{g}/\text{m}^3$ for the full pregnancy window—generally below the current regulatory level for annual mean PM_{2.5} in the United States of 12 $\mu\text{g}/\text{m}^3$ (<https://www.epa.gov/criteria-air-pollutants/naaqs-table>). Consideration of groups sensitive to the effects of PM_{2.5} (e.g., pregnant women, fetuses, children) in studies such as this one provide evidence to support strengthening the regulatory standard to protect these groups.¹⁵ Air pollution may be contributing to the burden of neonatal respiratory stress, including in places with ambient PM_{2.5} concentrations much higher than those reported in this study. Risks for adverse birth outcomes may be compounded for those who both live in high-exposure areas and do not have access to adequate hospital care during delivery, exacerbating perinatal health disparities. The results build on prior studies that, in totality, point to the need to educate policymakers, clinicians, and pregnant women regarding the effects of environmental risk factors, including PM_{2.5} exposure, on birth and neonatal health.

References

1. Stieb DM, Chen L, Eshoul M, Judek S. 2012. Ambient air pollution, birth weight and preterm birth: a systematic review and meta-analysis. *Environ Res* 117:100–111, PMID: 22726801, <https://doi.org/10.1016/j.envres.2012.05.007>.
2. Bekkar B, Pacheco S, Basu R, DeNicola N. 2020. Association of air pollution and heat exposure with preterm birth, low birth weight, and stillbirth in the US: a systematic review. *JAMA Netw Open* 3(6):e208243, PMID: 32556259, <https://doi.org/10.1001/jamanetworkopen.2020.8243>.
3. Markopoulou P, Papanikolaou E, Analytis A, Zoumakis E, Siahianidou T. 2019. Preterm birth as a risk factor for metabolic syndrome and cardiovascular disease in adult life: a systematic review and meta-analysis. *J Pediatr* 210:69–80.e5, PMID: 30992219, <https://doi.org/10.1016/j.jpeds.2019.02.041>.
4. Moster D, Lie RT, Markestad T. 2008. Long-term medical and social consequences of preterm birth. *N Engl J Med* 359(3):262–273, PMID: 18635431, <https://doi.org/10.1056/NEJMoa0706475>.

Address correspondence to Sheela Sathyanarayana. Email: Sheela.Sathyanarayana@seattlechildrens.org

The authors declare they have no conflicts of interest related to this work to disclose.

Received 9 November 2023; Revised 13 December 2023; Accepted 18 December 2023; Published 25 January 2024.

Note to readers with disabilities: *EHP* strives to ensure that all journal content is accessible to all readers. However, some figures and Supplemental Material published in *EHP* articles may not conform to 508 standards due to the complexity of the information being presented. If you need assistance accessing journal content, please contact ehpsubmissions@niehs.nih.gov. Our staff will work with you to assess and meet your accessibility needs within 3 working days.

5. Stewart DL, Barfield WD, Committee on Fetus and Newborn. 2019. Updates on an at-risk population: late-preterm and early-term infants. *Pediatrics* 144(5): e20192760, PMID: [31636141](#), <https://doi.org/10.1542/peds.2019-2760>.
6. Korten I, Ramsey K, Latzin P. 2017. Air pollution during pregnancy and lung development in the child. *Paediatr Respir Rev* 21:38–46, PMID: [27665510](#), <https://doi.org/10.1016/j.prrv.2016.08.008>.
7. Hehua Z, Qing C, Shanyan G, Qijun W, Yuhong Z. 2017. The impact of prenatal exposure to air pollution on childhood wheezing and asthma: a systematic review. *Environ Res* 159:519–530, PMID: [28888196](#), <https://doi.org/10.1016/j.envres.2017.08.038>.
8. Johnson M, Mazur L, Fisher M, Fraser WD, Sun L, Hystad P, et al. 2024. Prenatal exposure to air pollution and respiratory distress in term newborns: results from the MIREC prospective pregnancy cohort. *Environ Health Perspect* 132(1):017007, <https://doi.org/10.1289/EHP12880>.
9. Consortium on Safe Labor, Hibbard JU, Wilkins I, Sun L, Gregory K, Haberman S, et al. 2010. Respiratory morbidity in late preterm births. *JAMA* 304(4):419–425, PMID: [20664042](#), <https://doi.org/10.1001/jama.2010.1015>.
10. Lawn JE, Cousens S, Zupan J, Lancet Neonatal Survival Steering Team. 2005. 4 million neonatal deaths: when? Where? Why? *Lancet* 365(9462):891–900, PMID: [15752534](#), [https://doi.org/10.1016/S0140-6736\(05\)71048-5](https://doi.org/10.1016/S0140-6736(05)71048-5).
11. Ersch J, Roth-Kleiner M, Baeckert P, Bucher HU. 2007. Increasing incidence of respiratory distress in neonates. *Acta Paediatr* 96(11):1577–1581, PMID: [17937683](#), <https://doi.org/10.1111/j.1651-2227.2007.00440.x>.
12. Edwards MO, Kotecha SJ, Kotecha S. 2013. Respiratory distress of the term newborn infant. *Paediatr Respir Rev* 14(1):29–36, PMID: [23347658](#), <https://doi.org/10.1016/j.prrv.2012.02.002>.
13. García-Serna AM, Martín-Orozco E, Jiménez-Guerrero P, Hernández-Caselles T, Pérez-Fernández V, Cantero-Cano E, et al. 2022. Cytokine profiles in cord blood in relation to prenatal traffic-related air pollution: the NELA cohort. *Pediatr Allergy Immunol* 33(2):e13732, PMID: [35212052](#), <https://doi.org/10.1111/pai.13732>.
14. Rychlik KA, Secrest JR, Lau C, Pulczynski J, Zamora ML, Leal J, et al. 2019. In utero ultrafine particulate matter exposure causes offspring pulmonary immunosuppression. *Proc Natl Acad Sci USA* 116(9):3443–3448, PMID: [30808738](#), <https://doi.org/10.1073/pnas.1816103116>.
15. Tan S. 2023. Children's Health Protection Advisory Committee, Seattle and King County Public Health Department, to M. Regan, Administrator, U.S. Environmental Protection Agency. Climate Change Report. 28 August 2023. <https://www.regulations.gov/document/EPA-HQ-OA-2023-0030-0025> [accessed 6 November 2023].